ALBERTA HERITAGE FOUNDATION FOR MEDICAL RESEARCH

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AHFMR Mission

AHFMR supports a community of researchers who generate knowledge that improves the health and quality of life of Albertans and people throughout the world. AHFMR's long-term commitment is to fund basic, patient and health research based on international standards of excellence and carried out by new and established investigators and researchers in training.

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Views

Dr. David Hubel

"I remember my science teachers as being excellent. They made up in enthusiasm what they might have lacked in knowledge of the subject. My circumstances were advantageous for learning a lot about science at home because my father was a chemical engineer. The most outstanding of my teachers was my history teacher, who was incredible. She made me see that history is wonderful. I also give her credit for teaching me how to express myself properly in the language, an essential skill any young person needs, along with the ability to speak in public.

"There is always pressure on students to go into fields that have allure because of material compensation, but they should be allowed to follow what enthuses them. They need time to discover this, though. It is almost a truism to say if you could turn off 90% of TV, then kids would get bored. If they got bored enough, they would think up things to do that would interest them. If there had been TV around when I was young, I wouldn't have had the hobbies that eventually led me into science. It isn't necessarily the content of TV, [as much as] the time spent not doing something creative and interesting."

"There is a trend here [in American public schools] to amalgamate classes and make them big, ostensibly to justify other expenses, like having laboratories in schools. If I had to choose between a big class with a lab and a small class with no lab but where rapport can be established, I would choose the latter. I think it is a terrible mistake to have huge classes."

"Enthusiasm is an essential quality in teachers, along with a knowledge of the subject that is beyond the teaching level of it. In an ideal world, teachers should have some specialization in a subject that is more than a basic education degree. Teachers must be able to encourage and guide those students who are qualified to go further than the textbook. But the chief quality is enthusiasm. So much depends on teachers and their ability to interest kids in science."

Check "Reader Resources" on the inside back page for the Science Council Conference 2000 web site listing, where you can find information about public lectures to be given by the Nobel laureates.

Seeking answers from the immune system

This could have been another brain-drain story. Bright, young, highly educated Canadian goes off to do scientific research in the United States, and that's the last we see of her. But for Dr. Deborah Burshtyn, a Canadian immunologist who did her post-doctoral training at the U.S. National Institutes of Health, there were good reasons to come back to Canada. She joined the University of Alberta last year.

"One of the biggest attractions for me was the people in the U of A's Department of Medical Microbiology and Immunology," says Dr. Burshtyn. "There are a number of scientists here whose work is a really good fit with my research. This is an exciting place to be. The other major factor was research support."

Besides being an AHFMR Medical Scholar and a Canadian Institutes of Health Research (CIHR) Scholar, Dr. Burshtyn won the Peter Lougheed/CIHR Scholarship. This five-year award is given annually to the three top-rated Scholars in the CIHR program.

Now it's down to work, as Dr. Burshtyn extends the research she began for her Ph.D. Her interest lies in understanding how the immune system destroys tumour cells and cells infected with a virus.

The main immune system cells involved in killing infected cells are T cells and natural killer (NK) cells. The activation of T cells is fairly straightforward and well understood: Receptors on the T cell recognize an infected cell because they can bind to certain proteins on that cell. This activates the T cell, which then destroys the infected cell.

NK cells have an additional mechanism, since they not only activate, but inhibit as well: Certain receptors on NK cells are programmed to recognize proteins found on the outside of healthy cells. Once they find these proteins, an inhibition response blocks activation, and the NK cells do not destroy the healthy cell. However, on encountering an infected cell, there is no inhibition response, the T cell is activated, and proceeds to destroy its target.

"This is a very different way of activating an immune response," says Dr. Burshtyn. "While we know a bit about how this inhibition pathway starts, the subsequent steps are still a mystery. That's what we're looking at in my lab."

While this is basic scientific research, the results may one day have some very practical applications. For example, immunosuppressive therapies, such as those given to transplant patients, affect all the functions of the immune system throughout the body. "If we understood inhibition and activation of NK cells better, perhaps we could design therapies that are much more selective," adds Dr. Burshtyn. "We wouldn't have to knock out a person's entire immune system."

Right now, though, the focus is on experiments that will elucidate the inhibition pathway. "Science has come a long way in understanding the immune system on a molecular level," says Dr. Burshtyn. "At first, researchers concentrated on identifying the molecules involved in the immune system. Now, we're at the stage of defining what is happening."

It is painstaking work, but fascinating because of what Dr. Burshtyn calls the "data fix". "I really enjoy doing the experiments," she explains. "You think to yourself: Is it working this way or that? Then you design an experiment to test your ideas. Getting the answers is what drives me."

Dr. Deborah Burshtyn is a Heritage Scholar and Assistant Professor in the Department of Medical Microbiology and Immunology in the Faculty of Medicine and Dentistry at the University of Alberta. She is also a Peter Lougheed/CIHR Scholar.

What elements beyond biology, behaviour, and genetics affect our health? How do factors such as the work we do and the amount of money we earn determine how healthy we are? Answering these questions is the task of Dr. Kim Raine, a nutritionist and an AHFMR Health Scholar at the University of Alberta. Her challenge is to figure out how such social determinants work to influence health and well-being. Her research program focuses on poverty, gender issues, and the structure of the healthcare system.

Helping people lead healthier lives

Dr. Raine collects qualitative data in her work primarily by talking to people about their experiences. She conducts participatory research, in that the people who will potentially benefit by the research are directly involved in collecting the data. For one project on women's nutrition and body image, the nutritionist interviewed 47 women about their experiences and feelings about their bodies. She also asked them about aspects of their social environment that influenced the way they saw themselves, and how their body images were connected to eating patterns that might or might not be healthy. For another project, she collaborated with a community agency to find out the cost of healthy eating throughout Edmonton. In one very large project, Dr. Raine works with Alberta's Regional Health Authorities to find out their capacity to help communities prevent disease.

Promoting healthy living

The Heritage researcher's work goes a step beyond understanding how social determinants influence our health. She also tries to address factors that could make it easier for people to live healthier lives. Dr. Raine explains: "For example, if a person can't eat the foods we recommend because they aren't available in their environment or because the person can't afford to buy them, the health promotion strategy must address adequate income, affordability, and availability of healthy food." Out of the body-image project came a nutrition education program that is based on social change. "We're looking at how we can encourage women not only to cope with an environment that has media images of unrealistically thin models assaulting them every day, but to take action and actually demand that fashion magazines and other media not use anorexic models anymore," Dr. Raine comments. "We want to influence groups of people to work together to promote health in the broader community."

Dr. Kim Raine is an AHFMR Health Scholar and the recipient of a Health Research Fund grant administered by AHFMR for Alberta Health and Wellness. She is also an Associate Professor in the Centre for Health Promotion Studies at the University of Alberta.

'Determinants of health' is the collective label given to the multiple factors which are now thought to contribute to the health of populations. They include such things as people's biological endowment and individual responses, the social and physical environment in which they live, the economic conditions of their society, and the accessibility and quality of the health care system."

Health Canada, 1996

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You can't live without your liver.

It's essential to removing chemicals, poisons, and germs from your blood, making proteins and enzymes, regulating blood clotting, and absorbing fats and vitamins.

All in all, that's a pretty impressive "To Do" list.

Understanding

But for people with liver disease known as cirhosis, many of the items on the list are simply not getting done. Scar tissue has replaced normal, healthy tissue, blocking the flow of blood through the organ and preventing it from working as it should.

In North America, cirrhosis is the eighth leading cause of death by disease, killing about 25,000 people each year. Cirrhosis has many causes, but alcoholism, hepatitis C, and hepatitis B account for about 80% of all cases.

Because cirrhotic liver damage cannot be reversed, treatment focuses on preventing further progression and reducing complications. Loss of liver function affects the body in many ways including: jaundice; fluid accumulation in the legs and abdomen; toxin build-up in the blood and brain; and enlarged blood vessels which are prone to burst.

Alberta is fortunate to have AHFMR Senior Medical Scholar Dr. Sam Lee directing his talents to understanding liver disease. Born in Korea, raised in Newfoundland, and trained in Toronto and Paris, Dr. Lee came to the University of Calgary in 1988. "I had no western connection, but the Heritage Foundation offered great support for my research," he says. "I never would have come here otherwise."

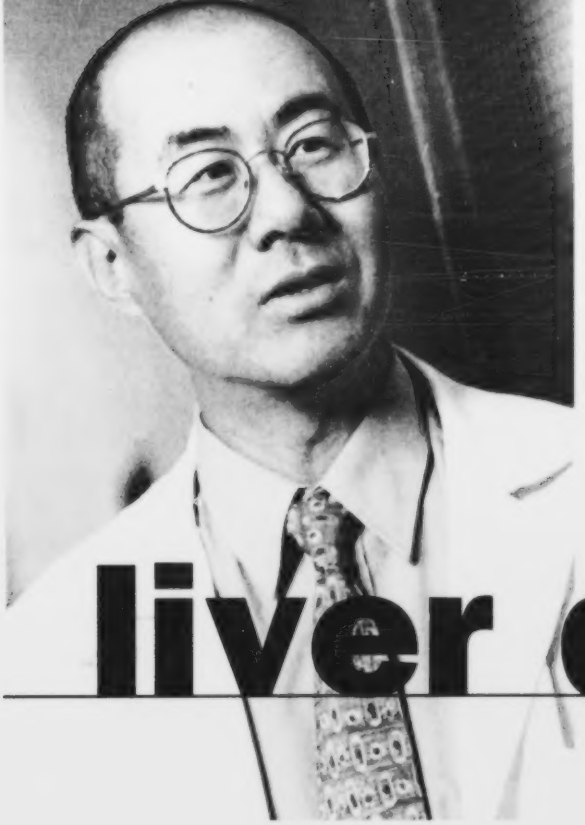
Dr. Lee, a hepatologist (a specialist in liver disease), is particularly interested in one of the most serious complications of cirrhosis: hyperdynamic circulation.

"Liver disease causes the circulation to go out of whack," he explains. "The heart pumps more blood and the blood vessels dilate. Because blood pressure drops, the kidneys think the body is in shock, so they conserve fluid. It's really a very peculiar situation."

Besides resulting in fluid accumulation, hyperdynamic circulation also affects the heart directly. When a person has hyperdynamic circulation, and an added strain is put on the heart—during exercise for example—the heart doesn't respond as it should. It can't contract more vigorously and pump faster. The condition is known as cirrhotic cardiomyopathy and it can lead to heart failure.

"Fifteen years ago, we thought cirrhotic cardiomyopathy was simply caused by excess alcohol consumption, not by something going on in the liver," says Dr. Lee. "Now we know differently." In fact, Dr. Lee's research has been key to illuminating the relationship between liver disease and heart function.

The problem seems to originate in the membrane of the heart muscle cells. In a heart affected by cirrhotic cardiomyopathy, the cell membrane is more rigid than normal. This rigidity impedes the function of a certain receptor, which is a necessary part of the signalling pathway that stimulates the heart.



Dr. Sam Lee had always planned on being a hepatologist, but not a researcher. A three-year research fellowship in France changed his mind. "I was fortunate to go to a very productive lab. I began to see research as a career. It's essential that our young, budding academics have the same kind of opportunity. We need to give them guidance so that they go to a place that will be good for their careers."

"Liver disease causes the circulation to go out of whack."

liver disease

"The pathway must be intact for the heart muscle to contract," explains Dr. Lee. "We don't really understand how cirrhosis actually affects the pathway. It could be that the increased level of lipids in the blood—a result of liver disease—alters the lipid structure of the cell membrane. Understanding the pathway is an active area of research."

One of the reasons for the interest in cirrhotic cardiomyopathy is increasing liver transplantations. (In Alberta last year, about 60 liver transplants were performed.) The operation stresses the cardiovascular system, and a person suffering from cirrhotic cardiomyopathy is particularly vulnerable to this stress. Cardiac failure has emerged as an important cause of illness and death in liver-transplant recipients.

"It may be possible to prevent the occurrence of cirrhotic cardiomyopathy by preventing the development of hyperdynamic circulation," says Dr. Lee. "This would be a great step forward for those suffering from liver disease."

But what controls hyperdynamic circulation? This question has led Dr. Lee to yet another organ: the brain. "The brain stem is the cardiovascular control centre," he explains. "In a normal person, if they lose blood, the brain stem sends a message to the heart to pump faster. In cirrhotic rats, the signalling seems to be missing. It's as if the circulation is turned on already, and can't be turned up any more."

Recent experiments on cirrhotic rats in Dr. Lee's lab have led to the discovery of certain compounds that can re-establish this signalling pathway. His team has found that by administering these compounds to rats, they can make the hyperdynamic circulation disappear. "It's very promising, but there's so much more to find out," adds Dr. Lee.

There are now a number of scientists around the world studying hyperdynamic circulation, but it wasn't always the case. Dr. Lee is a pioneer in both cirrhotic cardiomyopathy and neural control of hyperdynamic circulation. For years, these areas were largely ignored by researchers working on liver disease.

"I felt a bit like a voice in the wilderness," says Dr. Lee. "I was sometimes discouraged, but I have a thick skin. I never questioned why I was doing this research, I just wondered why others were blind to its value.

"Now that liver transplants have become so commonplace, heart problems have come to the fore. In the past five years, I've had a steady stream of requests for international speaking engagements. Before that, no one really paid any attention. It's good to see some eyes being opened."

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gut reactions

Could it be that we're too clean for our own good? One of the emerging theories behind the causes of immune diseases is that we live in a squeaky clean world where such staples as antibacterial soap and processed foods retard the development of robust immune systems. If a child's immune system doesn't wrestle with germs and develop antibodies to them, it may miss several key developmental stages. Later on, the immune system can go off the rails, either attacking the body's own tissues in a case of mistaken identity, or not turning off the attack after an invader is dead. The result can be one of an array of such immune diseases as diabetes, lupus, or inflammatory bowel disease.

Inflammatory bowel disease (IBD) is a term that includes Crohn's disease and ulcerative colitis, both caused by an as-yet-unknown combination of genes, faulty immune systems, and perhaps bacteria or viruses. Northern Alberta has one of the highest rates of incidence. Dr. Karen Madsen conducts basic research in IBD, focusing on Crohn's disease, an inflammatory condition that can affect any part of the digestive tract, from the mouth down. Crohn's is incurable and can relapse many times, rendering a person weak with pain, fever, maldigestion and malabsorption, sometimes intestinal blockages, and often serious side effects of the powerful drugs taken to keep the disease under control.

Scientists have shown that if mice with genes for IBD are raised in a bacteria-free environment, they do not contract IBD; those mice that are raised normally, however, do develop the disease. Another observation is that the IBD mice lack the protective "good" bacteria that coat the gut like a thick white paste, protecting the outside of cells. Given that there are 30 to 40 species of bacteria in the gut, both good and bad, the challenge is to discover which of the "bad" bacteria are linked to IBD.

Dr. Madsen is investigating the possibility of using protective "good" bacteria to prevent IBD. She comments: "When mice with the gene for IBD are treated from birth with a special concoction of protective bacteria, they don't get sick. However, if they are allowed to get sick, they get the disease for life." She has been using a special mix of protective bacteria that she plans to test on Crohn's patients in clinical trials at the University of Alberta.

In the meantime, Dr. Madsen is trying to work out the signalling that goes on between the protective bacteria, the epithelial cells lining the gut, and immune cells. She grew epithelial cells in a Petri

"Inflammatory bowel disease isn't life-threatening but sufferers have a diminished quality of life."

dish, added protective bacteria for a period of time, then took it away. When she later added Salmonella bacteria, the epithelial cells did not let the Salmonella invade, as if the protective mix had "vaccinated" them.

In another project, Dr. Madsen is exploring ways to stop the chronic inflammation that plagues Crohn's victims. The intestines of patients with Crohn's disease are very permeable; that is, the epithelial cells have spaces between them and no longer act as a barrier against bacteria. An enzyme called PARP, found in the nucleus of all cells, is activated by any kind of DNA damage. Once activated, PARP uses the energy that epithelial cells need to keep their protective membranes closed. If Dr. Madsen can stop PARP activity in epithelial cells, it would be key to helping the intestinal tract maintain its impermeability.

Testing "good" bacteria

The clinical trials referred to in the article are being run in conjunction with Dr. Richard Fedorak. Subjects for the trials will be selected from patients in the Division of Gastroenterology at the University of Alberta.

Macrophage cells signal the rest of the immune system to attack, and this signalling is preceded by an increase in PARP activity. This normally beneficial PARP activity is devastating in Crohn's patients, because the immune system treats the site of inflammation as a foreign invasion and attacks it, worsening the inflammation. If PARP could be stopped, the signals would not go out to the immune system, which would reduce inflammation and allow healing.

Dr. Madsen is investigating the use of a special chemical compound that appears to inhibit PARP in epithelial or macrophage cells.

"IBD isn't usually life-threatening, but sufferers have a very diminished quality of life. Once inflammation occurs, it's a vicious cycle," says Dr. Madsen. "My goal is to try and find the mechanisms to stop the cycle."

Dr. Karen Madsen is a Heritage Scholar and an Assistant Professor in the Division of gastroenterology at the University of Alberta. She is also funded by the Crohn's and Colitis Foundation of Canada.

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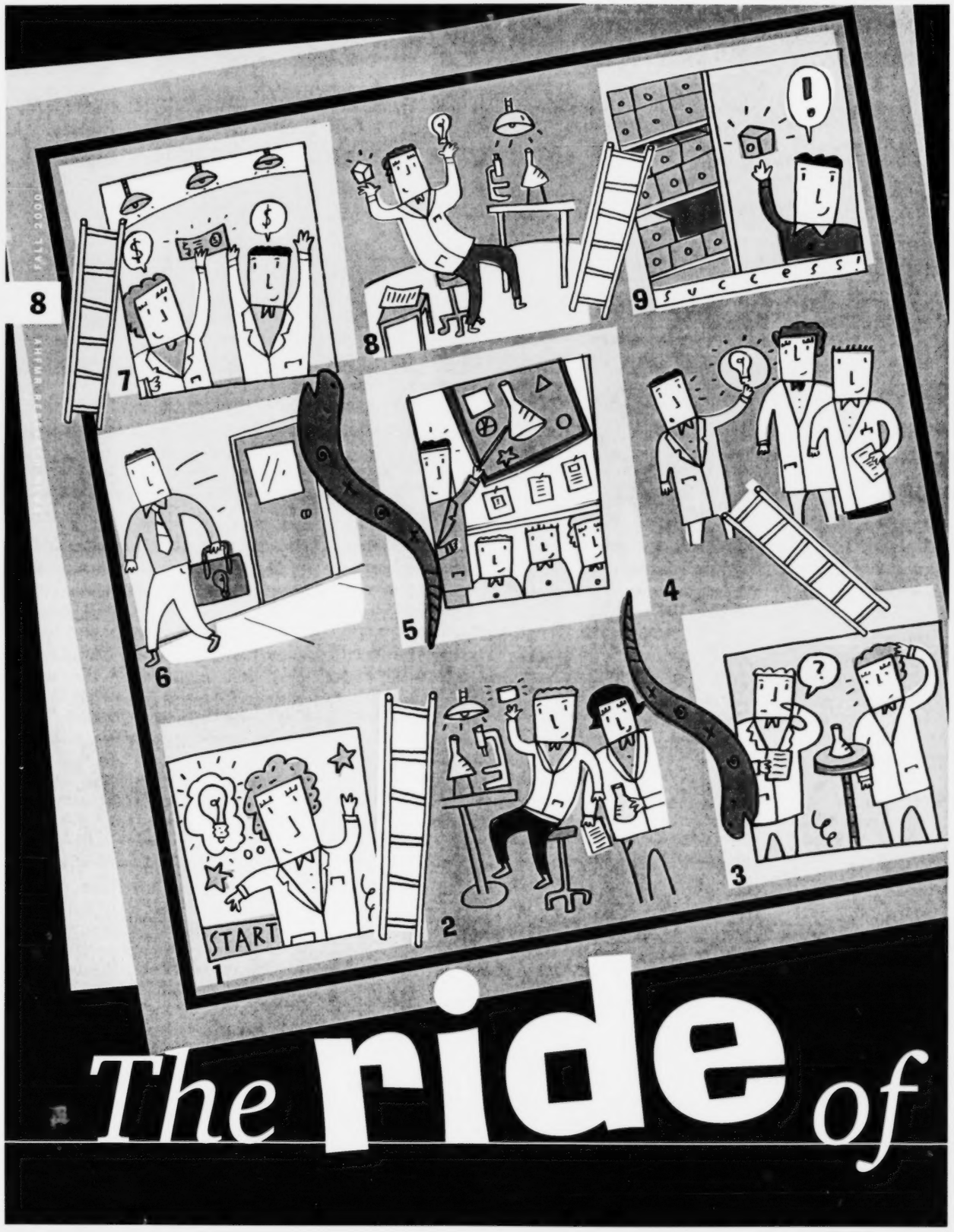
"IBD isn't usually life-threatening, but sufferers have a very diminished quality of life. Once inflammation occurs, it's a vicious cycle," says Dr. Madsen. "My goal is to try and find the mechanisms to stop the cycle."

Dr. Karen Madsen is a Heritage Scholar and an Assistant Professor in the Division of gastroenterology at the University of Alberta. She is also funded by the Crohn's and Colitis Foundation of Canada.



The ride of their lives

Biotechnology is all about high risk, elusive profitability, and extreme volatility. Products often take 12 to 15 years to get to market; the sky-high valuations of companies can plummet in a few days; investors spook easily. Many companies simply don't survive the development process. It's not a business for the faint-hearted.

If you're a player in this sector, you may well end up with nothing. You had better be prepared to endure—maybe even enjoy—the ride.

Some Albertans are doing just that.

An Alberta history

Alberta's first major foray into biotech was Chembiomed Ltd. Established in 1977, the company aimed to commercialize the pioneering carbohydrate chemistry done by University of Alberta's Dr. Ray Lemieux. In short order, other biotech companies sprouted up. Some, like Biomira Inc., are still very active. Many of the others no longer exist.

In 1991, the research programs of Chembiomed were incorporated into the Alberta Research Council. The technology re-emerged in 1994 in Calgary-based SYNSORB Biotech Inc. The products made by this publicly traded pharmaceutical company target bacterial toxins in the gastrointestinal tract. One of its lead products, SYNSORB Pk, was rushed to Ontario during the Walkerton crisis, when the town's drinking water was contaminated with E. coli bacteria. SYNSORB Pk is the only drug available to prevent the serious complications associated with E. coli infections. (It is now in the late stages of clinical trials, not yet available commercially.)

SYNSORB's President and CEO, Dr. David Cox, is proud of how far SYNSORB has come in a relatively short time. "We now have two products in Phase III clinical trials," he says. "I'm not sure there's another Canadian company that can make this claim."

However, there is a lot of biotech activity at earlier stages of development. In Alberta, researchers in molecular biology, genetics, agriculture, and many other disciplines have discovered and identified compounds with commercial potential. Alberta universities have licensed technologies and created new companies. Success is far from certain, as most of these start-ups struggle to move innovative compounds out of the lab. These new companies will require more money, which may be difficult to obtain. There's also the risk that their innovations may be rendered obsolete by newer, better technology.

Vital beginnings

Whatever the potential problems, SYNSORB's David Cox says the creation of new companies based on research and development (R & D) done in Alberta is vital. "Alberta has spent more than its fair share on R & D, yet I think it's fair to say that we've seen less than a fair share of the benefits. One way to change this is to encourage the commercialization of R & D, and to nurture the creation and growth of small companies.

"For this, we need an enterprise culture in the life sciences. This culture already exists in the oil and gas sector, but there aren't many Canadians eager to start companies in the life sciences.

"I think this is partly a consequence of the way we train our scientists. Our young people have to see the opportunities in biotechnology. They have to be shown that you can still love your science and make a buck."

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"Our young people have to see the opportunities in biotechnology."

Climbing to the top

That appears to be the way things are working out for Dr. Matthew Coffey. The 29-year-old from Medicine Hat finished his Ph.D. in 1998 and is vice president of a small biotech company. In only a few years, Coffey has racked up experience in finance, investor relations, clinical-trial management, and business planning that would be the envy of most 40-year-olds.

Sipping a latte in a Calgary café and talking about his career to date, Coffey's laid-back nature belies ambition and intelligence.

After completing his undergraduate degree at the University of Calgary, Coffey was keen to do graduate work. "If it hadn't been for the scholarship I received from AHFMR, I doubt I'd be sitting here today talking about biotechnology," says Coffey.

Coffey enrolled in the Ph.D. program and teamed up with Jim Strong, who was finishing his Ph.D. in Dr. Patrick Lee's lab. They were both extending Lee's ground-breaking work on the cancer-killing capability of a common virus called the reovirus, which is able to kill certain cancer cells by replicating inside them.

"We began to wonder about commercializing the technology," says Coffey. But there would be patents to file, development work to do, and a company to manage. Lee called on AHFMR's Technology Commercialization (TC) Program for help.

A helping hand from Heritage

"Patrick knew commercialization would not be easy," notes Linda Humphreys, Director of the TC Program. "He had a good grasp of all the issues and pitfalls, and was particularly concerned about fairness to his students and the University. The drive of Patrick's team to advance the technology forced them to work through the issues."

Start-up funds from the TC Program helped create a company–Oncolytics Biotech Inc.–and get patents in place. While the patents were important, Lee and Coffey knew they were no guarantee of commercial success. (Strong had gone on to do a medical degree, and wasn't involved with the company.) Oncolytics had to be managed, but none of the principals had any demonstrated business expertise.

A pioneer in the field

Enter Dr. Brad Thompson. A key figure in Alberta biotechnology, Thompson got his first taste of private-sector biotech while he was working as a scientist at the Alberta Research Council. He was there when Chembiomed was added to the ARC's portfolio. He was also heavily involved in the ARC's attempts to expand its biotechnology services into the United States. Thompson eventually became CEO of the Chembiomed spin-off company, SYNSORB Biotech.

While at SYNSORB, Oncolytics caught Thompson's eye. So much so that he is now the company's President and CEO. With Thompson's business know-how, Oncolytics has achieved some major milestones. Its product, REOLYSIN, is now in Phase I clinical trials at the Tom Baker Cancer Centre in Calgary. The company has also secured $23 million in financing, enough to cover Phase II trials, clinical studies on the veterinary use of REOLYSIN, and operating expenses.

"Brad made a real difference to Oncolytics," says Patrick Lee. "He has the contacts, he made the connections we needed. I simply couldn't have done what he has.

"Another thing is that Brad is in Alberta. We didn't have to get on a plane to meet him. We could get together over coffee. People say that distance doesn't matter anymore. Well, for this kind of interaction, I think it really matters."

ABOVE CENTRE: DR. MATTHEW COFFEY AND DR. BRAD THOMPSON

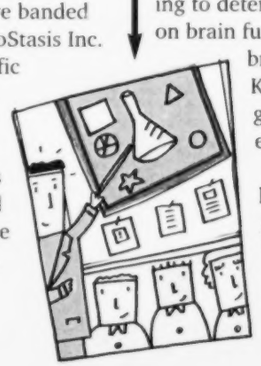
With Thompson on board at Oncolytics, Coffey shifted from the lab into the company. "There's still lots for me to learn," he adds. "As REOLYSIN moves into Phase II trials, it will be vitally important for us to expose the company to the US market. Exciting times are ahead."

On the trail of a new idea

You'll hear the same enthusiasm over at the University of Calgary. There, a promising collaboration is doing research that could make it possible to design drugs that promote natural brain repair.

The work involves Drs. Derrick Rancourt and Sam Weiss, and the University of Lethbridge researcher Dr. Bryan Kolb. The three professors have banded together to form a company called NeuroStasis Inc.

Their story begins with a major scientific discovery eight years ago. It had long been thought that there were no stem cells (the basic, primitive cells from which all other cells evolve) in the brains of mammals. That's why brain injury and disease was so serious–there was no hope for growing new cells. In 1992, however, Sam Weiss and his colleague, Brent Reynolds, stunned the scientific community by finding stem cells in the adult mouse brain. When stimulated by growth chemicals, the stem cells produced new neurons, the nerve cells that wire the brain. The two scientists founded NeuroSpheres Ltd. to commercialize this work.



In 1995 the company was sold to Ciba Geigy (now Novartis) and in 1998, its Calgary research operations were closed. Weiss returned to full-time university duties; he was instrumental in creating the new Genes & Development Research Group at the University of Calgary. But if Weiss thought his days in commercialization were over, he was soon proved wrong.

In 1995, scientist Derrick Rancourt had been recruited to the University of Calgary. Specializing in the genetics of embryonic stem cells, Rancourt and his team were amazingly productive. They identified about 300 new genes that are expressed in the embryonic nervous system. It was a joint proposal for equipment to continue this work that brought Rancourt in close contact with Weiss.

The two scientists saw exciting potential in collaboration. Research has found that stem cells in the central nervous system can grow into the three major types of brain cells. The discovery opens up the possibility of repairing brain injury and treating conditions such as Alzheimer's and Parkinson's.

"I wanted to use genes to cure neurological disorders," says Rancourt. "Sam suggested we focus on neural stem cells. Our intent is to find targets for drugs that will promote brain repair."

Rancourt and Weiss recognized that a necessary element would be animal testing to determine effects on brain function. They brought in Bryan Kolb, a neuropsychologist who is a world leader in evaluating the behaviour of rats and mice.

"Between Bryan, Derrick, and myself, we have a unique combination of talent that goes from genes to behaviour," explains Weiss. "This is the kind of comprehensive program that pharmaceutical companies

LOWER RIGHT (L TO R): DR. SAM WEISS AND DR. DERRICK RANCOURT

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would die for. But first we had to show we could make it work. We needed money to do the research that would prove our point. I knew granting agencies wouldn't be that interested. I thought maybe we could get private funding. But I needed a reality check."

Weiss's next step was to call AHFMR's Linda Humphreys. "Linda came down to Calgary for a meeting. She understood what we were trying to do and outlined how AHFMR might assist us."

After a thorough review, AHFMR helped with funding a marketing study, a patent strategy, and a business plan. Things looked good, but a major question still remained: Where would the operating capital for research come from? About the same time, Weiss and Rancourt met with Brad Thompson, who had agreed to review the NeuroStasis business plan.

"I had hoped to come out of the meeting not totally devastated by what Brad said," recalls Weiss. "As a scientist, your training is such that you're always expecting someone to shoot your ideas down. That's the way science is. It was a shock, then, to encounter such enthusiasm from Brad."

Not only enthusiasm; commitment as well. Thompson agreed to sit on the NeuroStasis board and help the company raise the needed capital. In short order, Thompson attracted an Alberta investor, allowing NeuroStasis to rev up its R & D program.

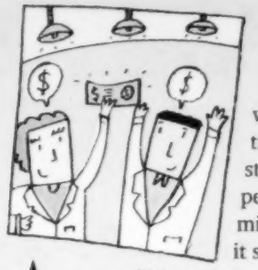
"Our pace is frenetic. We've hired new people, and data is emerging rapidly. We really hit the ground running," says Weiss.

This ability to move quickly has impressed both Rancourt and Weiss, who in the past have been frustrated by the time it takes to move a commercial idea ahead within the university system.

"Alberta is working toward having a critical mass of people in the biosciences," notes Weiss. "The intellectual property is emerging from scholars. There are dozens of commercial opportunities floating around our universities. But so much is being lost because of a 'wait and see' attitude.

"We need more people like Brad Thompson. People who can identify potential, protect that technology, and find the money to develop it. Even before a professor thinks about writing a paper."

So what exactly is Brad Thompson's magic? Well, don't expect him to tell you. For Thompson, the



whole process of commercialization is straightforward. The stumbling blocks that other people always bring up, he dismisses. In fact, Thompson makes it sound easy.

Money and risk

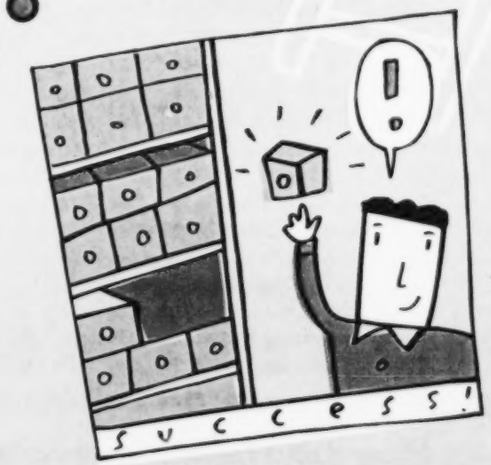
Money? "Alberta is a great place to raise money," says Thompson. "Since July 1999, the companies I've been involved in have had $80 million invested; $60 million of that came from Alberta. The finance community in Alberta is receptive to new ideas. But there's no special treatment for the biosciences, nor should there be. We need to offer returns that are comparable to sectors such as Internet, software, oil and gas."

Risk? There are risks in any undertaking, says Thompson, who doesn't believe that biotech has the corner on uncertainty. "It's all a matter of perspective. When people talk about the risks in a start-up company, they're really just talking about a different kind of risk."

Maybe success in biotech isn't only about great technology or access to capital. Perhaps confidence and insight are key to enduring and even enjoying the biotech ride. Alberta's scientific entrepreneurs and their fledgling biotech companies will surely find out.

Dr. Sam Weiss is a Heritage Medical Scientist in the Department of Cell Biology and Anatomy and the Department of Pharmacology and Therapeutics in the Faculty of Medicine at the University of Calgary.

Dr. Derrick Rancourt is a Heritage Scholar and Assistant Professor in the Department of Oncology and the Department of Biochemistry and Molecular Biology in the Faculty of Medicine at the University of Calgary.



Improving the odds for transplant success

"A kidney transplant is a wonderful gift."

But there are implications. The longer it takes to get an organ from a donor to a recipient, the greater the damage to the organ. Called ischemia, the damage results from loss of oxygen to the organ, and can cause both short-term and long-term problems for the transplant recipient.

"Kidneys that have been deprived of blood for some time are often slow to begin working after transplantation," explains University of Calgary nephrologist (kidney specialist) Dr. Lee Anne Tibbles. "They also tend to have more problems with rejection and don't last as long.

"Patients have asked me 'Why did my transplant fail?' or 'Why didn't my new kidney last longer?' These are questions that intrigue me. A kidney transplant is a wonderful gift. So what can we do to give each patient the very best chance for long-term success?"

Dr. Tibbles' hunch is that the answer lies in a chain of events that is triggered when blood begins to circulate again (reperfuse) through a transplanted kidney. She has been working on a series of pathways that transmit information within cells, using signal regulators called protein kinases. These proteins relay information between one another to propagate messages around the cell.

The stress of ischemia and reperfusion activates certain pathways in the cells that line blood vessels in the kidney. Signals cascade through these pathways, resulting in the production of adhesion molecules. These molecules line the kidney's blood vessels and act as little flags that attract the attention of the body's immune system. White blood cells attach themselves to the adhesion molecules, then move into the surrounding tissue, destroying it and harming the transplanted kidney.

"Immunosuppressive drugs can help, but they aren't the complete answer," notes Dr. Tibbles. "Immunosuppression only targets the white blood cells, and the therapy itself has complications such as increased risk of infection.

"My intent is to try to inhibit the signalling process so that adhesion molecules are not produced."

To do this, Dr. Tibbles' research team is exploring the use of a number of inhibitors. These inhibitors are similar to the protein kinases that make up the pathways; however, they cannot pass on a signal, and they also prevent any kinases around them from transmitting a signal.

The next step for the team is physiological and biochemical testing of the inhibitors in real kidney cells and blood-vessel-lining cells in test-tube studies. Once the most promising inhibitors are identified, they can proceed to animal testing.

"There's a lot more work to do," says Dr. Tibbles. "There are probably several pathways stimulated by ischemia, and we certainly don't know everything about them. But the field is growing fast and researchers are uncovering new information all the time. Our findings should be applicable not only to kidney transplants but other organs, and perhaps autoimmune diseases as well.

"It's interesting how this work started with a clinical problem, then moved to the molecular level. We're now about to start physiological testing. Hopefully, one day we'll be able to apply the results to transplant recipients."

Dr. Lee Anne Tibbles is a Heritage Clinical Investigator in the Faculty of Medicine at the University of Calgary. She also receives funding from the Kidney Foundation of Canada.

Improving the odds for transplant success

It seems remarkable that a donor in Ontario can provide a kidney to a recipient in Alberta, yet few people give much thought to the implications of that kidney's long plane flight, packed on ice.

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Many of us may remember the high-pitched noise that televisions used to make as we turned them on. Perhaps we credited technological advances with having refined the noise out of existence. However, children can confirm that the noise is still there. What has changed is that age and years of exposure to noise have diminished our hearing range.

Making sound waves in the brain

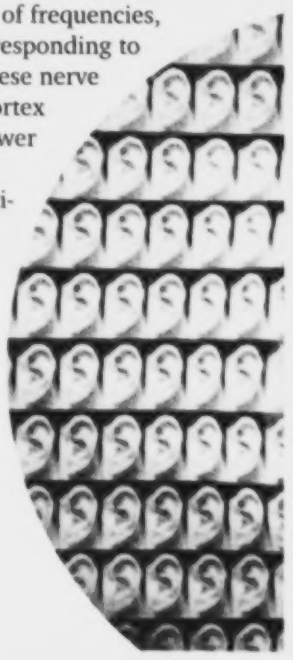
There is plenty of evidence linking hearing loss and episodic exposure to loud noise. The damage occurs in the cochlea, the delicate, fluid-filled canals in the inner ear. Hearing aids can overcome part of the hearing loss, but there is more than the ear involved. Dr. Jos Eggermont's research shows that the part of the brain responsible for hearing—the auditory cortex—changes as a result of that damage in the inner ear. With normal hearing, nerve cells in the cortex respond to sound waves from a full range of sounds. However, after noise-induced hearing loss, the response properties of large groups of these cortical neurons appear to change.

Part of Dr. Eggermont's work involves mapping the areas where the cortex is activated by sound frequencies.

Frequency is measured in a unit called a hertz (Hz), which represents one cycle of sound waves per second. Most speech frequencies, for example, range between 100 and 3000 Hz. In humans, the optimum frequency range for sound perception is around 3000 Hz. A whistle is between 4000 and 5000 Hz, Sounds above that are inaudible to most adults, but children can usually hear higher frequencies than their parents, such as the television's high-pitched signal, because they haven't yet suffered hearing loss due to noise exposure.

In cases of inner-ear hearing loss, Dr. Eggermont has observed that nerve cells in the cortex, which normally respond to upper ranges of frequencies, are more active. But they are not responding to the higher frequencies. Instead, these nerve cells in the damaged part of the cortex now respond to a narrower and lower range of hearing frequencies, normally the responsibility of the cortical area bordering it. With many more nerve cells devoted to a narrow and lower frequency range, the sounds within the range are now magnified and may sound abnormally loud.

During silence, this "reorganized" brain area appears more active than the normal cortex. This corresponds to a condition



called tinnitus, affecting about 5% of the population, where sufferers hear noises (ringing, buzzing, hissing) of undetermined origin. Scientists think that some forms of tinnitus are a by-product of the cortical reorganization that results from noise damage to the inner ear. In fact, Dr. Eggermont and his colleagues have demonstrated that spontaneous nerve-cell activity is much more extensive in the reorganized part of the cortex.

The cortex is also responsible for speech perception—that is, the way the brain codes the complex sounds of speech into meaning in the brain. Hearing loss affects speech perception dramatically. Two people with the same amount of hearing loss may have very different speech perception, resulting from different changes in their auditory cortex, which may explain why some people are helped by a hearing aid while others are not.

Dr. Eggermont is part of a research team that will compare the coding of complex sounds in a normal cortex with that in a hearing-damaged cortex. By recording sound-evoked brain activity during MRI (magnetic resonance imaging), the group will study brain changes in people who have suffered a sudden hearing loss on one side.

If a person with normal hearing is stimulated in one ear, corresponding brain activity occurs in the side of the cortex farthest away from the ear, so the right side of the cortex responds to the left ear, and vice versa. Brain activity occurs as well, at a lesser level, in the cortex nearest the stimulated ear, but the difference between the two levels of brain activity is disproportionate. In patients with one-sided hearing loss, however, there is an increase of brain activity on the cortical side that responds to the deaf ear over the course of about two years after the deafness occurred, showing that an adult cortex can still change. Whether this is an advantage or not is yet to be determined.

"If a child becomes completely deaf at age two, the part of the cortex responsible for hearing becomes frozen in time and stops developing."

Dr. Eggermont also has had the opportunity to study other effects on cortical development at the House Ear Institute, a Los Angeles centre for cochlear implants.

Cortical development in humans takes about 15 years. "If a child becomes completely deaf at age two, the part of the cortex responsible for hearing becomes frozen in time and stops developing," says Dr. Eggermont. "If the same child gets a cochlear implant, say at age six, the cortex picks up where it left off and continues developing as if nothing happened, although it lags behind. We recently found that if the period between becoming deaf and the application of the cochlear implant is too long, certain aspects of this development never happen. Hearing with the implant then becomes affected under difficult conditions, such as when several people are talking at the same time. We've noticed similar brain changes in adults who have received implants after going deaf as an adult." He has also observed that people who do well with their implant show the greatest amount of cortical development and change.

One of Dr. Eggermont's previous research interests, one that may seem strange to the lay person, was the study of oto-acoustic emissions—the sounds that come out of the healthy ear. It has become common practice to measure these sounds in gauging hearing loss and the need for hearing aids. Two of Dr. Eggermont's colleagues, Dr. David Brown (a former Ph.D. student) and Dr. Joseph Dort, recently received an Alberta Health and Wellness grant to conduct extensive screening of hearing in Alberta newborns by comparing oto-acoustic emissions and auditory brain-stem responses.

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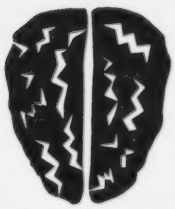

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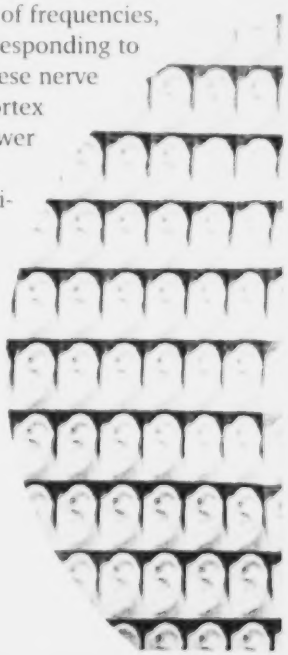

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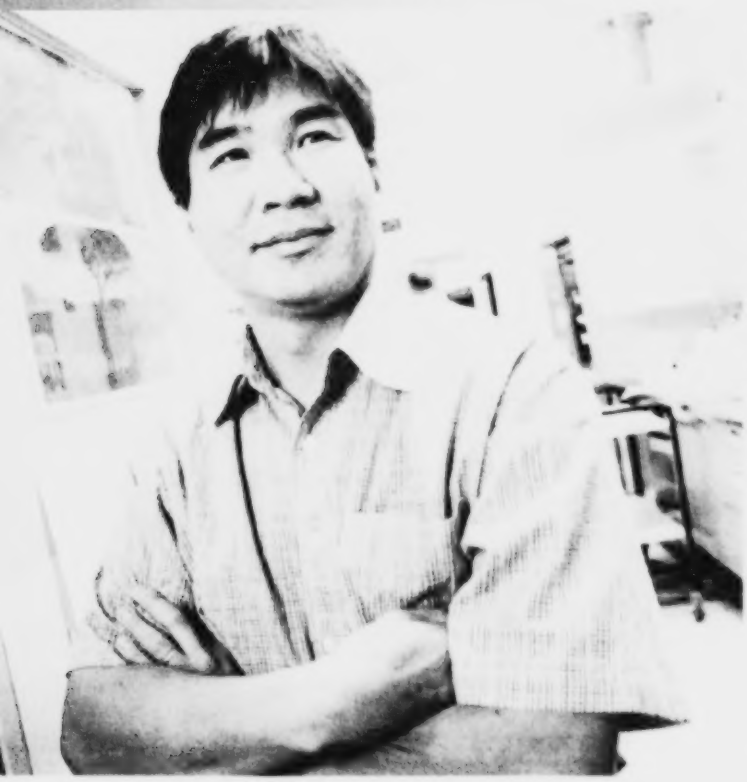
Dr. Jos Eggermont is a Heritage Medical Scientist, and Professor in the Department of Physiology and Biophysics, and the Department of Psychology at the University of Calgary. He also holds the Campbell McLaurin Chair for Hearing Deficiencies.

Seeking new treatments for a puzzling disease

To be diagnosed with multiple sclerosis (MS) is to find yourself on ground that is shifting under your feet—and to know that even if the shifting stops, it may well resume with a vengeance. One out of every 500 Albertans lives on this uncertain ground. For every man with MS, there are two women who have the disease, and the average age at diagnosis is 30. In a province with a young population like Alberta's, MS touches many lives.

"Probably 50 percent of us know someone or are related to someone who has MS," says neurologist Dr. Luanne Metz, Director of the Multiple Sclerosis Clinic at Calgary's Foothills Hospital and a member of the University of Calgary's MS Research Team. "The burden MS creates for people who have the disease, for their families, for the healthcare system, and for community resources is very, very heavy."

Physicians and scientists are all too aware that what they have to offer those who suffer from MS is limited. Current treatments include glatiramer acetate (Copaxone) and interferon-beta (Avonex, Betaseron, and Rebif). If started early enough, all of these drugs delay the permanent neurological injury that occurs with the disease. None of them, however, will cure MS or stop its progress.



The challenge for MS researchers is not just to develop brand new drugs—an enormously costly and time-consuming undertaking—but also to understand how the existing drugs work and how those mechanisms can be improved. "Understanding how drugs work in MS is very important in that it leads not only to a better understanding of what drugs are targeting, but it also helps to unravel some of the causes of what is happening in the disease," says Dr. V. Wee Yong, an AHFMR Senior Scholar and a colleague of Dr. Metz's on the MS Research Team. "Our aim is to help boost the efficacy of currently available drugs and, in so doing, to discover new therapeutics."

Dr. Yong and his colleagues at the University of Calgary are optimistic that they will soon be able to add another treatment to the MS medicine chest. Dr. Yong has been working with matrix metalloproteinases (MMPs), a family of proteins associated with inflammation in the central nervous system, and with the class of drugs known as matrix metalloproteinase inhibitors, which curb the effects of the proteins. "MMPs," he says, "are the bad guys in MS."

Interferon-beta is an MMP inhibitor that works by decreasing the amount of MMPs that leukocytes (white blood cells) produce, a discovery first published by the Yong laboratory. Copaxone works through a different mechanism: "We know that interferon-beta affects MMPs in one manner, and we are looking at drugs that are already in use which affect MMPs by other mechanisms," Dr. Yong explains. "If we can find other drugs with MMP-inhibitory activity, then we can use them to supplement the effects of the existing MS drugs. We'll be able to launch a multi-pronged attack on MMPs using interferon-beta and the second MMP inhibitor. Because these drugs are already on the market and have a proven safety record, it will be easier to get approval for their clinical use in MS patients. We have a candidate that has shown promise in animal models."

How MS drugs work

A colleague describes Dr. Wee Yong as "an international star" at understanding the mechanisms of the drugs used to treat multiple sclerosis. Indeed, as a consultant for Teva Pharmaceutical Industries, an Israeli company, he regularly packs his bags and heads off to explain to neurologists around North America exactly what those drugs do on a cellular level.

Like interferon-beta and glatiramer acetate (Copaxone), the current mainstays of MS treatment, the drug that Dr. Yong and the MS research team at the University of Calgary are hoping to bring to clinical trials in the next year acts as an inhibitor to the family of proteins known as matrix metalloproteinases (MMPs).

"MMPs act on a number of levels," Dr. Yong explains. "They help leukocytes [white blood cells] to traffic into the brain, and they produce inflammation when the cells go into the central nervous system. MMPs can also harm components within the brain, and there is evidence that they can degrade myelin and kill neurons. An MMP inhibitor would reduce the infiltration of leukocytes into the brain, curb the neuro-inflammation, and negate the toxicity of MMPs."

The MS team hopes to have clinical trials underway in about a year. To bring the drug to a Phase I trial, they have applied for funding through a new Canadian Institutes of Health Research (CIHR) grant competition for interdisciplinary teams of health researchers. The team is one of only 32 out of 179 groups—and the only MS research team—to have proceeded to the next stage of the highly competitive and lucrative awards. The winning teams, which will be announced in January 2001, will receive an estimated $1 million per year over a five-year period.

When the trials get underway, Dr. Metz is certain there will be no shortage of willing participants. "What we're trying to do is see if we can make the effect of current treatments better by combination therapy." However, even if that doesn't prove to be the case, showing that this drug is effective on its own—as this one was in the animal model—will also be important.

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"If we can show that combination therapy is more effective, we will be adding little to the current cost of treatment; whereas, if we show that this drug alone has similar efficacy and safety to current treatments, then the cost of treatment could be more in the range of $1,000 per patient per year," she says. "Greater therapeutic effectiveness, as we are hoping for from combination therapy, would then mean that patients get greater clinical benefits, and Alberta Health and Wellness gets more benefit for every dollar spent. On the other hand, a less expensive, equivalent therapy would mean that we could treat more people, and there would be more money available for other treatments. Even if we can show that this treatment is as good as the other treatments, then, at the very least, we can save money." rn

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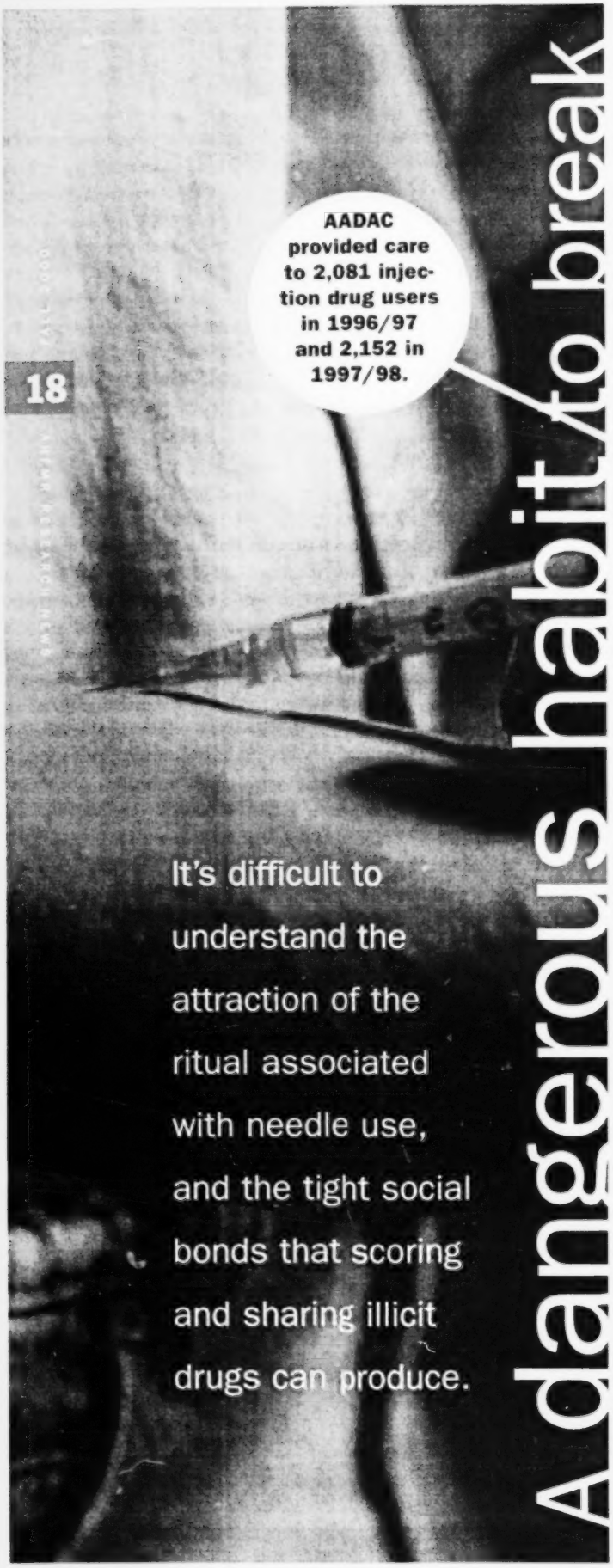
A dangerous habit to break



AADAC provided care to 2,081 injection drug users in 1996/97 and 2,152 in 1997/98.

It's difficult to understand the attraction of the ritual associated with needle use, and the tight social bonds that scoring and sharing illicit drugs can produce.

Until now, it's also been tough to get a complete picture of the health, social, and legal impact of injection drug use in Edmonton. As part of a research program on addiction and mental health, Dr. Cameron Wild is hitting city streets to talk to injection drug users about their habit, their health, and the high-risk behaviours they may be practising in order to get high. His new, two-year Health Research Fund project will help shed light on injection drug use (IDU), a growing problem in Edmonton. Increases in hepatitis C rates and emergency-room visits indicate that IDU is widespread. A conservative estimate of the number of injection drug users in the Capital Health Authority is 4,000, but that number may be considerably underestimated.

Edmonton's Streetworks outreach program exchanges approximately 550,000 clean needles and distributes 150,000 condoms yearly.

Part of the reason health-care providers don't have a better handle on the scope of the problem is that information about IDU being collected by various agencies is not integrated into a common database.

"The difficulty is that different agencies collect different information about this issue and house it in different locations and in different formats and oftentimes don't communicate it to each other," Dr. Wild notes. Connecting the data is an integral part of the project. "We urgently need to integrate data, so useful information can be disseminated and so we can study the health promotion needs of this population. That is what this research is all about."

Local programs provided by Streetworks, the Capital Health Authority, and the Alberta Alcohol and Drug Abuse Commission (AADAC) connect with people who inject drugs through outreach and treatment programs. But it is thought that the majority of users never seek help from outreach programs, nor do they access formal substance-abuse treatment. For this reason, Dr. Wild is conducting the study at street level. "We want to break through those barriers so we can gain an understanding of the health promotion needs of this group," he explains. "That will allow us to design strategies to help them promote their own health."

A research associate will work directly with Streetworks to distribute condoms and clean needles to users who want them. The research associate will record observations about each contact and try to build a rapport with this hard-to-reach population.

"This is the only way we'll get any insight into their lives," Dr. Wild comments. He hopes to get a sense of the users' histories of drug use (how they got to where they are). "From a scientific perspective, we need to have better information about the life course of injection drug users. We want to know how they started, what issues they think were pivotal in shaping where they are today, and how they use." He would also like to know what they consider safe and risky injection practices.

There are the pharmacological effects on the brain of injecting, but there are also highly potent sources of social influence and companionship in drug-using networks. Understanding the social dynamics of needle use will help Dr. Wild judge which factors could be modified to encourage safer drug use.

The research study is grounded in a harm-reduction framework. This perspective acknowledges that injection drug users may not be ready to stop using drugs. While total abstinence from drugs is desirable, it is unrealistic for many users. From a public health standpoint, it is important to intervene to reduce the health and social harm associated with IDU, employing a full range of strategies, from needle exchange to formal medical treatment.

In a four-month period from October 1998 to January 1999, there were 51 new mothers who reported they were using or had used illicit drugs.

"We want to know the users' perceptions of their health promotion needs," Dr. Wild says. "What do they see as barriers to accessing outreach and treatment programs? We know that we might not be able to find a magic bullet to stop injection drug use entirely, but the next best step is encouraging safer use."

The injection drug user population represented 4% of hospital emergency room visits in 1998.

Study results will provide a comprehensive and in-depth description of injection drug use in Edmonton and build bridges between the different sectors and agencies that serve the IDU population. Results will also help agencies such as Streetworks find better ways to access injection drug users and encourage them to safeguard their own health.

Dr. Cameron Wild is a Heritage Population Health Investigator and an Assistant Professor in the Department of Public Health Sciences and the Centre for Health Promotion Studies at the University of Alberta. His study of injection drug use in Edmonton is supported by the Health Research Fund, administered by AHFMR for Alberta Health and Wellness.

Harmful practices

Dirty needles contaminated with the blood of previous users, as well as contaminants in the drugs, can be deadly. Sharing needles is the major risk factor for HIV and can lead to such blood-borne infections as hepatitis B and C. These infections can cause illness and death among drug users, their sex partners—and even their children, through mother-to-infant transmission.

These health and social problems cost a lot. One Canadian study recently estimated that the combined health, social, and economic cost associated with injection drug use in Canada is about $49,000 per user per year. At the national level, conservative estimates are that illicit use of narcotics cost over $1 billion in 1992 alone. These costs are linked to lost productivity, direct healthcare costs, and law enforcement.

AHFMR Media Fellowship

Enhancing the quality of science coverage and fostering relationships between the scientific community and the media is the philosophy behind the AHFMR's Media Fellowship program. The 12-week program provides opportunities for one medicine, science, or engineering student at the University of Calgary and the University of Alberta to spend a good part of their summer working as reporters, researchers, or production assistants with a television or radio station, or major newspaper in Alberta. Sara Binder, a Calgary medical student, shares her experiences as a 2000 Media Fellow.

My experience as a Heritage Media Fellow working at CBC Radio has been wonderful. As a final year medical student, I have been contemplating ways to use my medical degree. My "dream job" would be to work as a talk-show host covering health related material. So when the Alberta Heritage Foundation for Medical Research posted the Media Fellowship position it seemed a perfect way to try out a combination of my interests, training, and skills.

On my second day on the job I had the opportunity to cover a story on the high rate of Sudden Infant Death Syndrome in Alberta's Aboriginal population. Over the next forty-eight hours I conducted interviews, put together my first script, a news clip, and a voiced news piece that aired on the morning news. I was ready to tackle anything after that "trial by fire"!

In subsequent weeks I worked on a number of fascinating stories. One that stands out involved a man suffering from diabetes. He is blind and close to needing dialysis for kidney failure as a result of his disease. Although dialysis units exist in Lethbridge and Fort McLeod, there isn't one in Cardston where he lives. The man is unable to drive because of his medical condition. Because his wife is the sole supporter for their family she is also unable to drive him two hours to Lethbridge for a five-hour session three times a week. What impressed me most about this story was the emotion and controversy it provoked, and the knowledge that there are so many stories out there that illustrate medical and health care issues.

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As the recipient of an AHFMR Media Fellowship Sara's Edmonton colleague **Laura Mireau** spent a busy summer working with a health and medicine reporter at ITV News. The enterprising student says she was challenged by the opportunity to attend news conferences, conduct interviews, and write scripts and stories that aired on the nightly news. Laura is currently pursuing a MSc in Immunology at the University of Alberta.

Working at CBC has given me the opportunity to talk to people from all walks of life about issues as diverse as sewage, million dollar deals with Nike, and the tragic death of a baby from SIDS. In addition to learning some of the technical aspects of broadcasting, I developed great respect for my colleagues. Their insight into spotting newsworthy issues and portraying them in a thought-provoking way is remarkable.

Thank you AHFMR for the great opportunity!

Sara Binder is currently completing her M.D. at the University of Calgary.

reader resources

Research Views: Dr. David Hubel and the ATA Science Council Conference

For more information on the Alberta Teachers' Association Science Council Conference 2000, please go to http://www.shep.net/conference2000/

Helping people lead healthier lives

Find out more about health promotion on the Health Canada website at: www.hc-sc.gc.ca/english/promo.htm

Understanding liver disease

Medline Plus Health Information (a service of the National Library of Medicine, USA)
http://medlineplus.adam.com/ency/article/000205.htm

Canadian Liver Foundation
http://www.liver.ca/docs/19980727_1.html

Gut reactions

For more information about IBD, call the Crohn's and Colitis Foundation of Canada in Edmonton at (780) 455-3319

In Calgary at (403) 266-2342
In Lethbridge at (403) 328-6868

Web site: http://www.ccfc.ca/

American Gastroenterological Society
http://www.gastro.org/ibd.html

The ride of their lives

SYNSORB
http://www.synsorb.com/

Oncolytics web site
http://www.oncolyticsbiotech.com/

Making sound waves in the brain

Resources for hearing impairment information:
Centers for Disease Control and Prevention
Developmental Disabilities Branch
http://www.cdc.gov/nceh/cddh/dd/ddhi.htm

KidsHealth
http://kidshealth.org/kid/health_problems/hearing_impairment_p2.html

Seeking treatments for a puzzling disease

Multiple Sclerosis Society of Canada (Calgary Chapter)
238, 2116 - 27 Avenue NE
Calgary, AB T2E 7A6

Multiple Sclerosis Society of Canada (Alberta Division)
10104 - 79 Street
Edmonton, AB T6A 3G3

Multiple Sclerosis Society of Canada (National Office)
250 Bloor Street East, Suite 1000
Toronto, ON M4W 3P9

Web page:
Multiple Sclerosis Society of Canada
www.mssoc.ca

A dangerous habit to break

Alberta Alcohol and Drug Abuse Commission
http://www.gov.ab.ca/aadac/

Canadian Centre on Substance Abuse
www.ccsa.ca

HIV InSite
http://hivinsite.ucsf.edu/

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Here's to your health in 2001!

AHFMR has produced a 2001 wall calendar as its report to the community. Colourful, informative, with original photos and fascinating images from research, and a large size calendar section for noting appointments, the AHFMR 2001 calendar will help you chart your way through the first year of the century.

2001
ahfmr
ALBERTA HERITAGE FOUNDATION FOR MEDICAL RESEARCH

The AHFMR 2001 wall calendar is a calendar plus more. You'll read highlights about current Heritage research in a number of health fields including heart and stroke, neuroscience, diabetes, early childhood, and seniors' health. You'll learn about the many Heritage funding and services programs that have helped make our province a hot-spot for biomedical and health research. And if you are already a subscriber, we invite you to pass the free AHFMR Research News subscription card on to a friend. You can also check out the AHFMR web site for more about the highlighted research in the calendar.

If you would like to receive the free AHFRM 2001 calendar, please call us at **(780) 423-5727**, e-mail us at **postmaster@ahfmr.ab.ca** or write us at:

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